South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 3 December 1960 Volume 34 No. 49 Deel 34 Kaapstad, 3 Desember 1960

INTRAMUSCULAR IRON IN NON-EUROPEAN PREMATURE INFANTS

ROBERT McDONALD, M.D., D.C.H., *Department of Child Health, University of Cape Town*

Iron depletion is liable to occur in the premature infant before the age of 4 months and unless substantial amounts of iron are absorbed, iron deficiency anaemia is said to be inevitable.[1]

Oral preparations of iron may prevent this, but little iron is stored by the body and it has to be given continuously, for at least the first 12 months, if the haemoglobin is to be kept at a reasonably high level. In a population which cannot be relied upon to carry out such prolonged treatment, this method will probably be unsuccessful.

Gaisford and Jennison[2] reported in 1955 on the intramuscular treatment of anaemic infants with the iron-dextran complex, 'imferon'. They also studied its prophylactic value against the anaemia of prematurity. Ten premature babies were each given 150 - 250 mg. of imferon, according to weight, at 3 weeks of age. The effect on the haemoglobin level was contrasted with that in 10 comparable babies not receiving iron supplements.

The authors found that at 16 weeks of age those babies who had received imferon had substantially higher haemoglobin levels than the control cases. The difference was maintained and increased until the 24th week, the last date reported in the article. At that time the average haemoglobin level in the imferon group was 82·2% (12·3 g. per 100 ml.), and in the untreated group 65·6% (9·9 g. per 100 ml.).

We decided in 1957 to repeat this trial on non-European babies in the premature unit at the Red Cross War Memorial Children's Hospital, Cape Town, to see if Gaisford's results could be reproduced. In this instance the babies were to be followed until the end of their first year to find out whether anaemia could be prevented, at any rate until this time.

Babies in this unit are born at home and are usually only accepted up to 48 hours after birth. In the group of infants to be discussed, most were within a few hours of birth, and all were less than 48 hours old when admitted, with the exception of one baby who was 4 days old and a pair of twins 7 days old when taken into hospital.

METHODS

Babies were selected by random sampling, either to receive imferon at 4 weeks of age, or to get no iron supplements. There were 38 cases in the first group and 35 in the second. The dosage of imferon was 150 mg. given as 50 mg. (1 ml.) daily for 3 successive days. A few babies were given 250 mg. spread over 5 days.

After these infants were discharged, their mothers were asked to bring them back to hospital for monthly follow-up examinations until the babies reached their first birthdays. At these visits the babies were weighed and their haemoglobin was estimated by the pathological service of the hospital, using a Klett-Summerson colorimeter. The infants were examined for, and enquiry made about, any infections since the previous visit. An approximate estimate of the physical and mental development was also recorded.

As far as could be determined, additional iron was not given to the babies after they had left hospital during the period of study, except in the case of the pair of twins who both received some oral preparation of iron for a while. As will be seen, this had little effect on their haemoglobin levels.

It was difficult to get mothers to bring their babies back regularly for a whole year and when it was decided to terminate the study, 2¼ years after its commencement, only 12 infants in each group had attended for the whole 12 month period required for the study. Of the rest some had died, but the great majority just failed to return for follow-up examinations, despite frequent letters from the social worker.

What follows, therefore, is a comparison between 12 babies who had not received iron supplements and 12 who had been given intramuscular iron. Nine of the latter had received 150 mg. of imferon and 3 had had 250 mg.

With so few cases, the results can do no more than indicate trends and should be considered in that light.

RESULTS

Table I gives the mean haemoglobin level in each group at intervals during the first 12 months of life. The levels are also shown in graphic form (Fig. 1).

TABLE I. MEAN HAEMOGLOBIN (G.%) DURING FIRST YEAR

Treatment	*Cases*	*Initial level*	*Level at age (in months)* 2	4	6	9	12
1. No iron ..	12	18·6	9·8	9·4	8·4	8·2	7·6
2. Imferon 150 mg. ..	9	18·7	9·3	10·0	9·9	9·7	9·2
3. Imferon 250 mg. ..	3	21·5	9·3	11·4	11·1	10·0	9·6
4. Combined imferon (lines 2 and 3) ..	12	19·4	9·3	10·4	10·2	9·7	9·3

Initial Hb. difference between 1 and 4 is not significant. $P > 0{\cdot}50$.
12-month Hb. difference between 1 and 4 is significant. $P < 0{\cdot}05$.

In the infants who received no supplementary iron there was a continuous fall in haemoglobin values throughout the year, reaching a mean level of 7·6 g. per 100 ml. at 12 months. Only 3 of the 12 babies had a haemoglobin of over 9 g. per 100 ml. at this time.

In the imferon-treated group, after a steep fall in haemoglobin at 2 months, which appears inevitable, there was a rise at 4 months, which was fairly well maintained

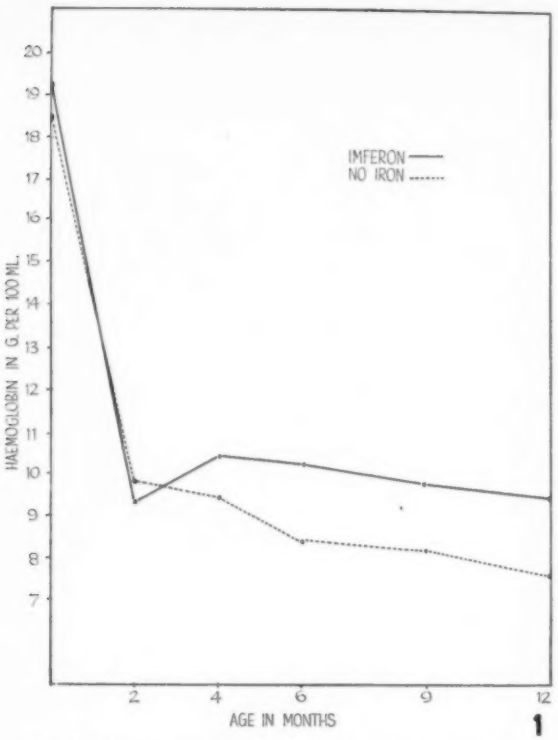

Fig. 1. Mean haemoglobin levels in premature infants. Twelve patients received imferon and 12 received no iron supplements.

to 6 months. Thereafter a gradual fall occurred to a mean level of 9·3 g. per 100 ml. at 1 year, but even at 4 months our figures were well below those reported by Gaisford and Jennison.[2]

The 3 babies who had been given 250 mg. of imferon were successive cases drawn for iron therapy and there was no selection on account of weight. By chance this was well distributed, being 2 lb. 15 oz., 3 lb. 7 oz., and 4 lb. 12 oz. respectively. Up to 6 months their mean haemoglobin figures were rather higher than those in the babies who had had 150 mg. of intramuscular iron. At 9 and 12 months, however, the figures in the 2 groups were not greatly different, and in the last line of Table I the haemoglobin levels for all 12 babies on imferon are combined.

In 9 additional patients in each group who attended only to the end of 4 months, the same haemoglobin differences at this time were found as in the groups completing the full 12 months' study period.

The figures in our pair of twins are of interest (Table II). The card drawn for the first twin was 'no iron' and the other was thereupon deliberately put into the imferon group in order to be able to compare results. At this time 250 mg. was being given and the second twin received that amount. The table illustrates once more the continuous fall in haemoglobin when no iron is given. By

TABLE II. TWIN BABIES

Patient		*Initial level*	*Level at age (in months)* 2	4	6	9	12
Charles J.	Hb. (g.%)	18·5	10·7	9·1	8·1	7·4	6·4
No iron	Wt. (lb./oz.)	3/3	5/3	9/13	12/11	15/8	18/3
Calvin J.	Hb. (g.%)	17·0	9·3	13·4	11·1	9·6	10·7
Imferon 250 mg.	Wt. (lb./oz.)	2/15	5/7	10/0	12/13	16/4	18/6

the end of the year there was a haemoglobin difference of 4 g. per 100 ml. in the 2 babies.

Mental defect, severe in 5 patients, appeared to be present in 12 of the 24 babies in the study. This was unrelated to the giving or withholding of iron but was found in those who had the lowest initial weights. All 12 weighed less than 4 lb. when first seen. This is in line with current opinion which suggests that the smallest babies at birth run the greatest risk of mental defect subsequently (Knobloch *et al.*,[3] Dann *et al.*,[4] Drillien[5]).

DISCUSSION

Other Studies

Hillborg and Nilsson[6] reported the use of imferon in premature babies in Sweden. As in our cases, those who were given imferon had a higher haemoglobin level at 4 months than those who had not received intramuscular iron. Their cases had a mean figure at that time of a little over 12 g. per 100 ml. and this level was maintained to the end of 12 months. Imferon dosage was 100 - 200 mg., most babies getting 150 mg. It is not possible to compare their cases where no imferon was given, with ours, as oral iron was given to the Swedish patients if the haemoglobin at any time fell below 11 g. per 100 ml. Their figures for 4 and 12 months remained in the region of 11 g. per 100 ml.

Hammond and Murphy[7] did a similar study in San Francisco. They used 100 mg. of imferon and from the third month found a highly significant difference in favour of the imferon-treated group (11·9 g. per 100 ml. compared with 10·5 g. per 100 ml.). This difference was maintained until 12 months and the authors' conclusion was that iron given in the neonatal period is able to prevent the late anaemia of prematurity. The article contains a comprehensive review of prematurity anaemia.

Reasons for Disappointing Results

In our patients on imferon the mean haemoglobin figure at 12 months was 9·3 g. per 100 ml. and in those who had not received iron supplements it was 7·6 g. per 100 ml. Despite this difference, which is a significant one, the figure of 9·3 g. per 100 ml. is disappointing and it cannot be said that the late anaemia of prematurity was entirely prevented. What is the likely reason for this?

Quantity of Imferon

Was too little imferon given? Gairdner[1] suggested that a baby needs about 245 mg. iron in his first year to supply growth requirements. Our 3 patients who were given 250 mg. imferon did not seem to do better than those who received only 150 mg. Hillborn and Nilsson[6] gave much the same dosage as we did and Hammond and

Murphy[7] smaller amounts, but in both groups their 12-month haemoglobin figures were well above ours.

Influence of Birth Weights

Did birth weights influence our figures? Were there, for instance, many babies in our imferon group with very low initial weights and did this affect the issue? Only 4 of our patients weighed 3 lb. or less and their mean haemoglobin at 12 months was 9·4 g. per 100 ml. At the other end of the scale, there was only 1 baby of over 4 lb. in the group and his 12-months' reading was 7·7 g. per 100 ml. Our initially smallest babies, therefore, did not have the lowest haemoglobin readings at 1 year.

It was noted, however, that whereas the group on imferon had a mean starting weight of 3 lb. 5 oz. compared with 3 lb. 10 oz. in the 'no iron' group, the mean weights of these groups at 1 year were 19 lb. 4 oz. and 16 lb. 6 oz. respectively. It is possible that the iron supplement may have improved the general metabolism of the recipients resulting in a better weight gain than that in the other group. The group on imferon presumably had the greatest increase in blood volume. Did those babies in the imferon group with the greatest weight gain have the lowest final haemoglobin levels? This certainly seemed to be the case in our 2 babies who had the greatest gain in weight. Each gained about 20 lb. in their first year and their final haemoglobin readings were 7·7 and 7·4 g. per 100 ml. On the other hand, 2 other babies gained just over 16 lb. each and their 12-month haemoglobin figures were both over 12 g. per 100 ml. The rest, who gained between 14 and 15 lb. had figures varying from 6·2 - 11·8 g. per 100 ml. with a mean of 9·4 g. per 100 ml. It did not seem, therefore, that weight gain kept the final haemoglobin figures down and, in any case, there were only 2 of the 12 babies who had a large weight gain. Admittedly there were only 12 cases, so perhaps one should not make too much of the above figures.

Infections

What about infection? Gaisford and Jennison[2] pointed out that this could nullify the effects of imferon and quoted one of their anaemic premature babies who had an initial rise of haemoglobin but, following an attack of bronchitis, this fell by 10%. Wintrobe,[8] on the contrary, has stated that infections lasting less than a month are not in most cases accompanied by significant anaemia. Lanzkowsky[9] found that when imferon was given to anaemic babies suffering from a coincident short-lived infection, such as pneumonia or gastro-enteritis, the iron was utilized and there was no interference with haemoglobin synthesis.

Most of our patients getting imferon had at least one infection and some several, during their first year. These infections were usually either gastro-enteritis or a respiratory infection and though some cases were severe, most were of quite short duration. Two of the 12 babies had only minor infections and it may be of significance that at the end of their first year they had the highest haemoglobin levels of all the babies, namely 12·4 and 11·8 g. per 100 ml. Each had received 150 mg. of imferon. Six more suffered from several infections, which, in nearly every instance, were followed by a drop in haemoglobin level. However, each of the 4 remaining patients had an infection between 9 and 10 months of age and in all of them the haemoglobin at 12 months was higher than at 9 months. It is difficult, therefore, to assess the effect of infection in our cases and it will require much larger numbers to determine the correct answer to this question.

Other Reasons

Is there any other reason to account for our figures not being higher at 12 months than they were? It is of interest to compare Lanzkowsky's[9] haemoglobin findings at 12 months in healthy full-term non-European children in Cape Town. The mean levels were 9·57 g. per 100 ml. in Coloured and 9·84 g. per 100 ml. in African babies. The corresponding figure for European babies in this series was 11·20 g. per 100 ml. The figures for non-European full-term babies at 12 months, who had received no particular iron supplements, are rather similar to the present series for premature infants given imferon. All that imferon seemed to have done was to bring the haemoglobin level of the premature babies up to a figure near to that of their untreated full-term counterparts, and emphasizes the anaemia which Lanzkowsky[9] has shown to be so common in non-European babies in Cape Town.

SUMMARY

Late prematurity anaemia is inevitable without the addition of iron supplements. In a population, which could not be relied upon to take oral preparations for a prolonged period, an attempt was made to prevent iron deficiency anaemia by giving intramuscular iron in the first weeks of life.

Although this resulted in better haemoglobin levels than in the untreated cases, the results were not very satisfactory as regards the prevention of anaemia, and even at 4 months our figures were well below those of other published series. It is difficult to be sure of the reason for this, particularly as it was not possible to get a large number of patients to cooperate in the study for a full 12 months.

The mean haemoglobin level at one year was similar to that found by Lanzkowsky[9] for healthy full-term non-European babies of the same age in Cape Town, and this may be a reflection of the general tendency to anaemia in the non-European children in this city.

The relation of infection to the drop in the haemoglobin level in this study was uncertain.

ADDENDUM

Is Imferon Dangerous?

Since this paper was completed a disturbing Leading Article now well known, appeared in the *British Medical Journal*.[10] In it the suggestion was made that imferon might be carcinogenic in man and the Journal recommended that the makers should withdraw the preparation.

The Leading Article was based on reports of experiments in rats and mice which were repeatedly injected with massive doses of imferon in the same site for a prolonged period.[11,12] A considerable number of the animals developed sarcomas.

Several letters to the Journal followed, notably one by Golberg[13] of the research department of the firm manufacturing imferon. He stated that, under similar conditions,

many preparations, including glucose, fructose and arachis oil were carcinogenic. He had been able to produce some sarcomas in mice by similar heavy dosage of imferon, but found only one in 50 hamsters so injected and none in rabits or guinea pigs. Golberg believed that, in the affected animals, repeated trauma and muscle necrosis at the site of the injections was more likely to be the cause of the sarcomas than was the imferon. He reported that skin sections from patients who had received subcutaneous injections of imferon showed almost total disappearance of iron after 23 days. Golberg maintained that although sarcomas could be produced in some laboratory animals, it was not logical to state that the same might therefore occur in man.

A further letter appeared in the *British Medical Journal* in June 1960[14] signed by 7 distinguished physicians; this carried considerable weight. The joint opinion of the signatories was that imferon, in the recommended dosage, carried a negligible risk and was probably less hazardous in other respects than intravenous iron or blood transfusion. Practitioners could, therefore, in their opinion, use the drug without fear.

The whole matter has aroused considerable interest and perturbation and has been reviewed in editorials in the Australian,[15] American,[16] and Canadian[17] medical press. The consensus of opinion of these journals was that, if given in the correct dosage and with circumspection, imferon is a valuable agent. All gave it as their opinion that the evidence for its toxicity was hardly convincing.

Here the matter rested until 27 August, when the *British Medical Journal* published an article by Robinson *et al.*[18] from Vancouver, Canada. This described a soft tissue sarcoma in the left deltoid region of a 74-year-old woman at the alleged site of imferon injections given 3 years previously. It was not claimed that the tumour was definitely caused by the imferon, especially since the sarcomas produced in rats by this method differed considerably in appearance. Nor could the authors say with certainty that the tumour in their patient was a primary one. Nevertheless it was suggested that imferon might possibly have been the carcinogenic agent.

The same issue of the Journal carried an Annotation[19] which reaffirmed the Journal's previous attitude, especially in view of the abovementioned case. A letter by Prof. A. Haddow in the same number[20] was also discussed in the Annotation. This letter pointed out that the risks of imferon would not be known for about 15 years from the time it was first used. In the meantime Haddow felt that it is not justifiable to assert that the preparation carries a negligible risk in man.

The latest word on the subject is a memorandum by Benger Laboratories Ltd., the manufacturers of imferon. This reports discussions between Dr. Golberg of Bengers and Dr. J. P. Smith of the Christie Hospital and Holt Radium Institute, Manchester, on the one hand, and the Canadian authors of the article mentioned above on the other. There would appear to be some doubt whether the patient did, in fact, receive imferon in her upper arm. Even if this was the case, the relation of imferon to the tumour is questioned, both because of the short latent period, and because of the histological appearance, which differed from that occurring in animals who had received excess dosage of intramuscular iron. It must, however, be stated that there may be a species difference in the histological appearance of the tumours.

Reports indicate that more than 1,000,000 persons have received imferon during the last few years. What are the harmful effects reported? One case of sarcoma in a 74-year-old patient with an unproved relationship.

It appears to me that the risk of imferon is little greater than is the case with other agents such as chloramphenicol, chlorpromazine and even penicillin. These drugs have not been withdrawn. Why then should imferon? Aplastic anaemia, liver disease and anaphylactic shock from the above drugs may all cause death, but the word 'cancer' probably still strikes more terror into the heart of the public than does any other. It is necessary, therefore, to have a balanced viewpoint in the case of imferon.

If there is a risk it should be a calculated one, and in this case it is to my mind so small as to be, in fact, negligible.

I have the authority of Prof. F. J. Ford, Head of the Department of Child Health at the University of Cape Town to state that it will be the Department's policy not to discourage the use of imferon provided it is not used indiscriminately. The chief indications for its use in paediatrics are the prevention of iron-deficiency anaemia in premature babies, and the treatment of this anaemia in infants and older children, but only in cases where the parents cannot be relied upon to give iron by mouth, or in the rare cases where oral iron cannot be tolerated.

As regards the efficacy of imferon there can be no doubt.

Thanks are tendered to Prof. F. J. Ford for helpful criticism and advice; to Dr. J. F. W. Mostert, Medical Superintendent of the Red Cross War Memorial Children's Hospital for permission to publish; to the Pathology Department of the Hospital for haemoglobin estimations; to Miss D. Barker for her valiant efforts to get the children back for follow-up studies, and to those mothers who so regularly brought their babies back for examination and without whose cooperation this study would not have been possible.

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South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

HYPERTENSION WITH STENOSIS OF THE RENAL ARTERY

In a previous issue of the *Journal*[1] we pointed out that the condition of narrowing of the renal artery has been added to the short list of curable hypertensive conditions. We also stated that 'although this condition is seen only rarely at present, it is possible that finer chemical and X-ray investigations might in due course lead to the diagnosis of more cases of this nature'.

An investigation of stenosis of the renal artery as a cause of hypertension has now been reported in the *British Medical Journal* by Brown, Owen, Peart, Robertson, and Sutton,[2] of St. Mary's Hospital, London. It is based on the study of twenty-two hypertensive patients in whom disease of the renal artery was proved or strongly suspected. Eleven of them were judged to be likely to benefit from operation. A routine examination by renal arteriography was made in a hundred and sixty cases of severe hypertension and a lesion of the renal artery was demonstrated in ten of them. In eight of the other twelve cases in the series of twenty-two, the narrowing of the renal artery was revealed at aortography performed for other reasons (usually disease of the aorta or iliacs). It is evident that renal-artery stenosis is not so rare a condition as has hitherto been supposed; it is being recognized in patients with high blood pressure with increasing frequency. The report also records highly favourable operative results in the cure of selected cases of hypertension caused by narrowing of the renal artery.

A wide variety of disorders may lead to this narrowing, but the most frequent cause is an atheromatous plaque, usually near the origin of the artery; and stenosis of the renal artery is to be suspected particularly in those hypertensive patients who have shown evidence of occlusive arterial disease elsewhere (e.g. intermittent claudication and coronary or cerebral arterial occlusion). However, hypertension with stenosis of the renal artery may occur without evidence of these other conditions, nor are these conditions rare in cases of hypertension without demonstrable renal-artery stenosis.

Cases of hypertension caused by stenosis of the renal artery show the usual signs of high blood pressure on clinical examination, but the only characteristic physical sign of the stenosis (not, however, present in every case) is a systolic bruit best heard near the mid-line of the abdomen, but occasionally confined to the costovertebral angle. In their twenty-two cases Brown *et al.*[2] record the murmur as present in ten. In every case operated the origin of the bruit in the renal artery was confirmed by direct auscultation of the artery; in some a thrill could be felt in the affected vessel. The murmur is usually abolished when the patency of the renal artery is restored.

The diagnosis of stenosis of the renal artery depends mainly on renal arteriography and ureteric catheterization studies. Arteriography is of great value in detecting the stenosis, but as it sometimes produces misleading appearances, and as a degree of stenosis may exist without serious effect on the kidney, the diagnosis should be confirmed by 'separate' renal-function studies. These were carried out in every case in the series recorded by Brown *et al.*[2] Intravenous pyelography, also, is often a valuable diagnostic test. All these procedures have their hazards and impose and ordeal on the patient. As Brown *et al.* state, none of them is to be undertaken lightly or by the inexperienced.

Three different techniques are available for renal arteriography, and each of these was used by Brown *et al.*[2] in different cases. They are (1) simple translumbar aortography, (2) aortography by percutaneous transfemoral catheterization by Seldinger's method,[3] and (3) percutaneus selective renal aortography (Ödman[4]) in which a radiopaque catheter is used. The choice of technique is largely a matter of personal preference.[2] Brown *et al.* favour a modification of Ödman's method devised by D. Sutton, by which excellent renal radiograms were regularly achieved.[2] In one of their cases a stenosis of the renal artery was diagnosed on the aortogram and further investigation showed that no such stenosis existed. 'In several cases . . . where a small artery was virtually occluded no such appearance was produced'.[2] Denny and Tinker,[6] of Johannesburg, found Seldinger's method most satisfactory.

The ureteric-catheterization studies described by these *et al.*[2] were carried out under spinal anaesthesia. Olivary-tipped ureteric catheters were passed *via* a cystoscope under X-ray control so that the tips lay in the respective renal pelves. A self-retaining catheter was left in the bladder so that any leak around the ureteric catheters should immediately become apparent, in which event they were adjusted to stop the leak or reduce it to a minimum; only in this way, the authors state, can accurate measurements of urine flow and clearances be made. This technique also avoids error arising from the movement of water and electrolytes across the walls of the ureter and bladder. After two satisfactory periods of urine collection during which the water load was maintained by oral replacement, 0·9 % sodium chloride containing inulin and sodium para-amino-hippurate (PAH) was infused at a constant rate into an arm vein and further periods of urine collection taken, after equilibration. (In some cases clearance of endogenous creatinine was measured instead of inulin clearance.) 'Apart from occasional discomfort to the patient, the procedure has been free from complications'.[2]

'The characteristic features of renal-artery stenosis . . . are, on the side with the lesion, diminished urine flow; lowered clearance of inulin, creatinine, and PAH; increased urinary concentrations of inulin, creatinine, and PAH; and usually, diminished urinary concentrations of sodium and chloride'.[2] These features derive from the fact that in the affected kidney there is a smaller volume of glomerular filtrate and greater reabsorption of water, sodium, and chloride, than in the opposite kidney. Brown *et al.*[2] record the results of their ureteric-catheterization studies in twelve proved or probable cases of renal-artery stenosis and nine controls. 'In all the probable and proved

cases the affected kidney produced less urine, with a higher concentration of PAH, inulin, and creatinine, and almost invariably a lower concentration of sodium and chloride, than its fellow. This pattern was never seen in the control group, where, if one kidney secreted less urine with a lower concentration of sodium than the other, a lower concentration of inulin and PAH was also found on that side'.[2]

As regards intravenous pyelography, the dyes used in excretion urography (sodium acetrizoate and diatrizoate) produce concentrations in the urine on the two sides which parallel inulin and creatinine concentrations. It was to be anticipated, therefore, that, provided the kidney had not been severely damaged, higher contrast and better visualization would be obtained on the side affected by renal-artery stenosis than on the normal side. In the series of twenty-two cases, better contrast on the side with stenosis was in fact found in seven. The authors[2] give a warning against a wrong impression that might be caused by this finding, viz. that the kidney with the normal artery is functionally impaired; 'there is a very real danger in these cases of the unaffected kidney being removed, with disastrous consequences'.[2] Failure to appreciate the meaning of this frequent result has led to a tendency to overstress the limitations of selective intravenous pyelography. It is often in fact, 'a most valuable diagnostic aid in renal-artery stenosis'.[2]

The surgical procedures that have been used to rectify stenosis of the renal artery include the following: thromboendarterectomy of the renal artery; anastomosis of the splenic artery to the distal renal artery (on the left side); reimplantation of the renal artery in a new site in the aorta; excision of the stenosis in cases where its situation in the renal artery enables this to be done, followed by end-to-end anastomosis; insertion of a bypass graft from the aorta to the distal renal artery; in cases of complete occlusion of the renal artery, as well as certain other cases, nephrectomy is the operation of choice.

In six of the cases in the series reported by Brown *et al.*,[2] which were submitted to nephrectomy or reconstructive surgery, the diagnosis of renal-artery stenosis 'was established beyond reasonable doubt'. In all six the hypertension was of a severe order, and in five of them the optic fundi showed bilateral exudate and haemorrhage (three showed papilloedema also). In these five the blood pressure became normal after the operation and the retinal abnormalities resolved, the heart became smaller, and the ECG signs of left ventricular strain disappeared. In the sixth case the hypertension was not cured. Three earlier cases in the series were operated on after less adequate investigation, and so the diagnosis was less surely established; in only one of these was the operation followed by remission of the blood pressure.

During operations for renal-artery stenosis it is important to measure the pressure in the renal artery distal to the stenosis by direct arterial puncture; 'unless a distinct pressure-drop across the site of the supposed obstruction is found, the diagnosis of a truly functional renal-artery stenosis should be strongly queried, whatever the arteriography appearances suggest'.[2] DeCamp and Birchall[5] hold that if there is no pressure-drop, renal-artery surgery should not be performed, but Brown *et al.*[2] consider that such cases merit further study. At the completion of a reconstructive operation the pressure in the renal artery distal to the site of the previous obstruction should also be measured; the operation can only be regarded as successful if this pressure has been restored to the level of the adjacent aortic pressure.[2]

In cases in which operation for stenosis of the renal artery is not considered to be indicated or has been unsuccessful, hypotensive drugs have been used with varying degrees of success.

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DIE BEHANDELING VAN DREIGENDE EN GEWOONTEMISKRAAM

Na 'n bepaling van die betekenis van die geskiedenis, simptome, en tekens van die pasiënt, en na skatting van hoe onvermydelik die proses van dreigende miskraam is (en hier mag die graad van servikale verwyding deurslaggewend wees), moet oor 'n program van behandeling besluit word. Stalworthy[1] sê: Die vier grondliggende beginsels by die hantering van miskraam is:

1. By elke miskraam is die behandeling onmiddellikspesifiek, *en* algemeen. Sodra die toestand suksesvol behandel is, moet pogings aangewend word om uit te vind waarom dit voorgekom het en planne moet beraam word om 'n herhaling te voorkom.

2. Wanneer 'n miskraam dreig, is die doel van behandeling om te voorkom dat dit onvermydelik word.

3. By alle ander gevalle van miskraam is die doel van die behandeling om te sorg dat die uterus leeg is, om bloeding te beheer, en om sepsis te voorkom.

4. Geen pasiënt moet van mediese sorg ontslaan word voordat dit seker is dat haar gesondheid heeltemal herstel het nie. 'n Ondersoek na 'n miskraam is net so belangrik soos 'n ondersoek voor die tyd. Stalworthy waarsku tereg dat chorion-karsinoom op miskraam mag volg.

Ons wil hier net die eerste twee beginsels bespreek. Rus in die bed oor 'n paar dae is raadsaam. Die waarde van 'n lang tydperk van rus in die bed is twyfelagtig en moontlik selfs nie raadsaam nie: trouens die gewone pligte kan binne 'n dag of twee hervat word. Gebruik van kalmeermiddels sal die ongerief van sametrekkings van die uterus verlig en die vrees vir vaginale bloeding verminder. Morfien en petidien is waardevol. Ons is nie bekend met 'n middel wat die sametrekkings van die uterus tot stilstand sal bring nie. Estrogeen en progesteroon word 'n geruime tyd lank al gebruik by die behandeling van hierdie toestand sonder oortuigende statistiese ondersteuning. Dit is nou bewys, nie net dat progesteroon in dosisse gebruik is wat glad te klein is om enige uitwerking te hê nie, maar ook dat genitale of mindere vermanlikingsanomaliteite by klein dogtertjies mag voorkom. Ten slotte kan die embrio alreeds dood wees teen die tyd dat die pasiënt die eerste keer gesien word en die gebruik van steroïdes mag die proses van miskraam verleng en teruggehoue miskraam by pasiënte veroorsaak in wie se gevalle die toestand nie korrek gediagnoseer is nie — iets wat selfs vir die deskundiges moeilik mag wees.

Daar is egter regverdiging vir die gebruik van steroïdes op grond van onlangse werk oor die bepaling van bloedprogesteroon (nie progesteroon in die urine nie, en die ontwikkeling en opkoms van die sterk en effektiewe norprogesteroon tipe van samestellings wat per mond geneem kan word, byvoorbeeld ‚noretidrel' en ‚noretidroon'. Dit is tot nog toe nie aangetoon dat hierdie produkte misvorming van die geslagsdele veroorsaak nie. Behandeling moet lank genoeg duur om die simptome te beheer, en moet opgehef word wanneer hulle opklaar. Behandeling moet natuurlik gestaak word as die beeld een word van onvermydelike of teruggehoue miskraam.

Pasiënte wat onderhewig is aan gewoontemiskraam moet natuurlik baie goed ondersoek word — selfs voor swangerskap. Afwykinge van die geslagsorgane kan dan vasgestel word, byvoorbeeld deur middel van 'n histerosalpingogram en, waar moontlik, reggestel word. Dieselfde geld vir dieettekorte, chroniese siekte, gebrekkige funksionering van die tiroïed, tekortkominge van die bloed, en onverenigbaarheid van die A-B-O bloedgroepe.

'n Belangstellende en simpatieke houding van die kant van die geneesheer is van onberekenbare waarde by die verligting van spanning en angs by hierdie pasiënte. Hierdie faktor behoort altyd 'n prominente plek in te neem by die behandeling van miskraam, aangesien daar nêrens in die hele gebied van mediese praktyk so baie onstabiele en emosioneel-versteurde pasiënte is nie as diegene wie se begeerte om kinders te hê onbevredig moet bly.[2] Dit is ook van belang dat psigiatriese hulp ingeroep moet word in die gevalle waar daar duidelike emosionele moeilikhede bestaan; so ook is die ondersteuning van die pasiënt se eie geneesheer van groot belang in hierdie gevalle.

Ons moet nog op drie verdere belangrike punte wys in verband met die behandeling van dreigende of gewoontemiskraam. Eerstens lyk dit of dit nodig is dat alle pasiënte wat onderhewig is aan vroeë dreigende miskraam, wat nog nie onvermydelik geword het nie, die voordeel behoort te kry van vroeë behandeling met die norprogesteroon tipe van produkte. Die aantal pasiënte by wie dreigende miskraam nog nie onvermydelik geword het nie, kan op hierdie manier gehelp word. Tweedens moet daarop gewys word dat nie alle vroeë bloeding die gevolg is van dreigende miskraam nie, aangesien ongeveer twintig persent van gevalle van swangerskap vroeë bloeding kry as gevolg van oorsake waaroor daar nog gespekuleer word. Derdens, dit is nie vasgestel dat die progesteroon tipe van produkte die fetus benadeel soos die suiwer progesteroonstowwe nie (wat volgens moderne standaarde in elk geval in dosisse gebruik word wat glad te klein is om swangerskap in stand te hou). 'n Mens hoef dus nie onnodig te vrees dat hierdie soort behandeling aanleiding sal gee tot die geboorte van 'n abnormale baba nie.

Laastens, kan die volgende belangrike prognostiese feit genoem word: die gegewens tot ons beskikking dui sterk daarop dat die kanse om 'n misvormde kind te hê nie vermeerder word in swangerskappe wat gekompliseer word deur dreigende miskrame nie, ook is daar nie bewys dat die behandeling van dreigende miskraam die voorkoms van aangebore gebreke verhoog nie.

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A CLINICAL TRIAL OF GUANETHIDINE IN HYPERTENSION

J. C. SHEE, M.D., M.R.C.P., D.T.M. & H., *Senior Honorary Consulting Physician, Bulawayo Hospital Group;* B. J. BALDACHIN, M.R.C.P.E., D.C.H., *Senior Physician, Mpilo Hospital;* and J. E. P. THOMAS, M.R.C.P., D.C.H., *Physician, Mpilo Hospital*

Guanethidine (Ismelin, Ciba Su- 5864) belongs to a new series of compounds synthesized in recent years by Mull *et al.*[1] in the Ciba Research Laboratories, Summit, New Jersey.

The hypotensive action of this drug was first described by Maxwell *et al.*;[2] and preliminary clinical reports by Page and Dustan,[3] and Frohlich and Freis[4] appeared in the USA in July and August 1959 respectively. The first British experience with the drug was published by Leishman *et al.*,[5] in December 1959. As these, and the few other clinical reports which have appeared, were generally favourable (Richardson and Wyso,[6] Jaquerod and Spuhler[7]) it was decided to conduct a clinical trial on African and European hypertensive patients in Central Africa.

CHEMISTRY AND PHARMACOLOGY

The clinical name of guanethidine is 2 - (octahydro - 1 - azocinyl) - ethyl - guandidine sulphate, and its structural formula is shown in Fig. 1.

Guanethidine is not chemically related to bretylium, but, generally speaking, has a similar action in that it appears to effect a specific blockade of the sympathetic nervous pathways without a corresponding blocking of the parasympathetic system, such as occurs with ganglion-blocking drugs like pentolinium and pempidine.[5]

Maxwell *et al.*,[8] showed that guanethidine profoundly lowered the arterial pressure of unanaesthetized neurogenic (amphetamine induced) and renal hypertensive dogs. It had a slight hypotensive action in the normotensive unanaesthetized and anaesthetized dog. It was likewise a potent antagonist of the pressor responses elicited by (*a*) bilateral occlusion of the carotid arteries, and (*b*) injection of high doses of amphetamine.

Within a period of 6 - 12 hours after its intravenous injection, the nictitating membranes and the arterial system

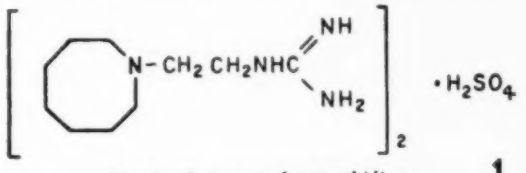

Fig. 1.—Structure of guanethidine. 1

become unresponsive to stimulation of efferent sympathetic fibres. During the same period the nictitating membranes and the arterial system are strongly responsive to injected noradrenaline. These effects last from 4 days to 3 weeks. Zaimis[9] produced evidence that adrenaline may be concerned in the 'recovery' process of a cell after activation. Then the changes produced by guanethidine, bretylium and certain other drugs on the 'recovery' mechanism could be explained as due to a 'displacement' of adrenaline from its points of attachment.

From the clinical aspect, the prolonged action of a single dose of the drug offers the advantage of infrequent dosage and smooth control of hypertension, without the daily oscillations in pressure found in other anti-hypertensive drugs. Unlike the ganglion-blocking drugs it does not produce parasympathetic side-effects such as dry mouth, constipation, and ileus; in fact, owing to the unopposed action of the parasympathetic, diarrhoea may be an annoying but not serious side-effect. It is readily controlled with propantheline bromide.[7]

METHOD, CLINICAL MATERIAL, AND RESULTS

At the start of the investigation all patients were admitted to hospital for full clinical assessment and to allow the effect of previous hypotensive drugs to wear off. As experience with the drug increased, a number were treated entirely as out-patients. All blood-pressure readings were taken first with the patient supine and, when the condition of the patient allowed it, after standing for 2 minutes. This latter precaution is important, because it was found that the full postural hypotensive effect of the drug was not evinced until that period of time had elapsed. In-patients had their blood-pressure readings taken twice daily, out-patients initially every 2 or 3 days, and later, when stabilized, at intervals as long as 4 weeks. When it seemed to be indicated, a diuretic of the benzothiadiazine group was given in addition. The material was drawn from private consultant practice, and from European and African hospital practice. A total of 23 patients, 12 of them European and 11 African, were considered to be sufficiently studied and documented for inclusion in this paper. The diagnoses of the group are set out in Table I.

TABLE I. DISTRIBUTION OF CASES

	No.
Essential hypertension	16
Malignant hypertension	3
Primary nephrosclerosis	1
Diabetic nephropathy	1
Chronic pyelonephritis	2
Total	23

TABLE II. RESULTS OF FOLLOW-UP STUDIES

Case	*Age*	*Sex*	*Race*	*Diagnosis*	*Pre-treatment blood pressure (mm. Hg)*	*Duration of treatment in weeks*	*Additional therapy*	*Final daily dose (mg.)*	*Final blood pressure*	*Side-effects*
1	43	F	Eur.	Malignant hypertension	L 286/160 S 240/130	12	B	60	L 212/120 S 152/104	Nil
2	44	M	Eur.	Essential hypertension	L 230/140 S 210/130	16	Nil	25	L 116/88 S 104/82	Failure of ejaculation. Postural hypotension
3	71	M	Eur.	Diabetic nephropathy	L 210/140 S 208/140	6	B Insulin	25	L 136/90 S 112/70	Postural hypotension
4	68	M	Eur.	Essential hypertension. CHF. Previous CT.	L 202/132 S 196/124	10	B	12½	L 142/72 S 122/60	Nil
5	62	M	Eur.	Essential hypertension	L 202/130 S 172/120	15	Prednisolone (for asthma)	60	L 176/110 S 158/110	Slight postural hypotension
6	52	M	Eur.	Essential hypertension	L 232/160 S 224/154	5	B	25	L 182/112 S 146/ 90	Nil
7	53	M	Eur.	Essential hypertension. Recent hemiplegia	L 264/168	12	B	50	L 184/106 S 178/102	Nasal congestion
8	46	F	Eur.	Nephrosclerosis	L 260/160 S 232/152	16	B	37½	L 222/128 S 192/106	Nil
9	83	F	Eur.	Essential hypertension	L 232/140	9	Nil	25	L 182/108 S 160/108	Nil
10	68	F	Eur.	Essential hypertension, angina pectoris	L 194/130	4	Nil	37½	L 168/104 S 142/ 90	Status anginosus (treatment discontinued)
11	62	F	Eur.	Essential hypertension. HE, DU + haematemesis	L 256/150	9	Ac	37½	L 182/90 S 146/84	Nil
12	62	F	Eur.	Essential hypertension. Recent hemiplegia	L 268/170	2	B	40	S 190/110	Nil
13	50	F	Afr.	Essential hypertension. CHF.	S 240/140	3	Nil	30	S 175/95	Nil
14	21	M	Afr.	Essential hypertension	S 175/145	5	Nil	40	S 120/90	Nil
15	55	M	Afr.	Nephrosclerosis	S 220/140	6	Nil	60	S 220/120	Nasal discomfort, mild diarrhoea
16	40	M	Afr.	Malignant hypertension	S 220/180	3	Nil	50	S 190/130	Nil
17	46	M	Afr.	Malignant hypertension	S 250/170	11	Nil	75	S 175/110	Postural hypotension + mild diarrhoea
18	28	M	Afr.	Essential hypertension	S 200/130	9	Nil	20	S 170/100	Impotence (temporary)
19	50	F	Afr.	Essential hypertension. CHF.	S 200/120	10	Nil	30	S 120/90	Nil
20	42	F	Afr.	Essential hypertension	S 210/140	5	Nil	40	S 165/105	Nil
21	30	F	Afr.	Chronic pyelonephritis	S 230/140	12	Nil	70	S 190/130	Nil
22	70	M	Afr.	Chronic pyelonephritis	S 200/140	4	Nil	20	S 150/90	Nil
23	55	F	Afr.	Essential hypertension	S 175/130	5	Nil	40	S 140/100	Nil

L = lying; S = standing; B = diuretic of benzothiadiazine group; CHF = congestive heart failure; CT = coronary thrombosis; Ac = Anticholinergic; HE = hypertensive encephalopathy; DU = duodenal ulcer. **N.B.** Ages of African patients were approximate.

Patients were followed up for periods ranging from 2 to 16 weeks, and the results of the investigations are set out in Table II.

DISCUSSION

Although this is a limited trial, the results obtained to date (Table II) indicate that the use of guanethidine in the treatment of hypertension is a method of considerable promise.

Effects on Blood Pressure

It will be seen from the results tabulated that the drug caused a fall in blood pressure to satisfactory levels in 21 out of the 23 cases treated. The 2 exceptions were both African patients. Case 15 is an African male aged approximately 55, who is being treated as an out-patient with weekly increases of dosage as indicated. It is probable that the effective maintenance dosage has not yet been reached. Case 21 is an African female, aged 30, who showed little response to a dosage of 20 mg. daily, but who has not been followed up any further, since she defaulted from the trial.

The effect of the drug is cumulative, a fact which was demonstrated by the gradual fall of blood pressure in the standing position over several days on a constant dosage. For this reason the initial dosage should be low, and an increase should be made at intervals of several days, as indicated by blood-pressure readings. In this trial, the initial dosage in most cases was 10 mg. daily, given in the morning. This was increased by 10 mg. at a time, at intervals of 4 - 7 days, until a dosage was reached at which a satisfactory level of blood pressure was recorded. In most cases (18 out of the 23 cases) this maintenance dosage was between 25 and 60 mg. daily. The remaining 5 cases required doses that were either smaller or larger than those within this range.

Although the fall in blood pressure was largely a postural effect, there was in some cases an appreciable fall in the lying position as well after a few weeks' treatment. This phenomenon was more noticeable in the European than in the African patients (Table II). (Unfortunately no accurate records were kept of blood pressure in the recumbent position in African patients). This effect on blood pressure in the lying position has been noted in previous papers.[4,7]

The prolonged smooth effect of guanethidine, permitting its administration in a single daily dose which may be taken at any time during the day, is of considerable advantage. This is particularly so in dealing with African patients, where simplicity of dosage and administration is most desirable in a condition which requires treatment for an indefinite period. We did not find any evidence of appreciable diurnal variations in blood pressure, as may occur with ganglion-blocking agents, e.g. bretylium.

Effect on Electrocardiograph

As the blood pressure fell and was maintained at a near normal level, the ECGs of several of the patients showed considerable or almost complete reversal of the patterns of LV strain and hypertrophy which were present before treatment. In the short period of study T-wave changes reverted before the high-voltage pattern was reduced. More prolonged study is necessary before it will be known whether the ECGs, in any cases, will revert completely to a normal pattern.

The improvement in the ECG seems to be roughly parallel to the degree of objective improvement in the ocular fundi. Fig. 2 shows the left-pectoral leads of 2 patients who showed considerable improvement after a short period of treatment.

Side-Effects

Various side-effects have been described. These include diarrhoea, bradycardia, parotid tenderness, nasal obstruction, tremor, fluid retention, breathlessness, weakness, tiredness and depression, and failure of ejaculation.[10] Most of these are similar to the side-effects which have been found with bretylium.

The side-effects have not usually been severe. In particular, the parasympatholytic side-effects of the ganglion-blocking agents have been absent. Case 17 (African male, aged 46), who has been described in an earlier paper,[11] could not be treated with ganglion-blocking agents because of severe side-effects. He developed tolerance to bretylium, but has been well-controlled on guanethidine. Another

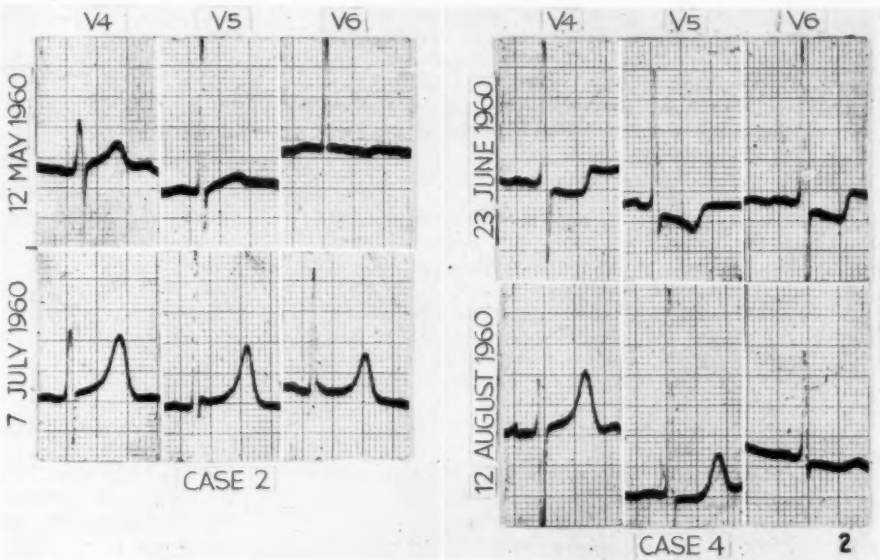

Fig. 2. Left pectoral leads from cases 2 and 4 showing considerable improvement in the T-wave changes after short periods of treatment.

patient, case 8 (European female, aged 46), was also treated originally with bretylium, but this was abandoned owing to episodes of severe postural hypotension. She has been controlled satisfactorily with guanethidine — without side-effects.

Faintness and dizziness as a consequence of postural hypotension was the most common side-effect in our series, but this was never severe enough to effect continuation of treatment. It was always relieved by adopting the recumbent position for a short while. Dollery *et al.*[10] have noted that this symptom is aggravated by exercise, and this is a factor which will undoubtedly need to be taken into consideration in future, particularly in the treatment of manual labourers.

Mild diarrhoea, which amounted only to the passage of 3 or 4 semi-formed stools per day, occurred in 2 African patients, who were receiving fairly high doses of the drug. The symptom was not considered severe enough to warrant special treatment. Where indicated, it may be controlled by kaolin, small doses of pempidine,[11] or anticholinergic drugs.

Nasal congestion and discomfort occurred in 2 patients, and failure of ejaculation in 1. Impotence occurred in an African male aged 28; this was a temporary symptom, lasting for 2 weeks only, and may not have been due to treatment. Treatment had to be discontinued in 1 patient because of persistent angina pectoris (case 10).

Tolerance to the drug has not yet developed in any case treated. Indeed, it is possible that the opposite may occur. Cases 2 and 3 in our series had their original maintenance dosage reduced, because of increasing symptoms of postural hypotension.

Addition of Benzothiadiazine Diuretics

Seven patients were given oral diuretics of the benzothiadiazine group. In most of them it was possible to reduce the daily dose of guanethidine for maintenance requirements, thereby reducing the incidence of side-effects. This finding agrees with a previous report on the use of diuretics in combination with guanethidine[7] and may be an advantage in patients in whom side-effects are troublesome. Combined therapy was not used in African patients, because it was felt that a multiplicity of tablets would lead to confusion.

Effect on Renal Function

Insufficient investigation has been carried out to comment fully on this factor, but no deterioration of renal function has been noted in any of our patients.

SUMMARY AND CONCLUSIONS

23 patients suffering from severe hypertension have been treated with guanethidine, a new adrenergic-blocking agent. The drug was used in both European and African patients. A satisfactory smooth reduction of blood pressure was obtained in 21 of the 23 cases, with minimal side-effects.

The main advantages of guanethidine in the treatment of hypertension are: ease of administration, lack of development of tolerance, freedom from parasympatholytic side-effects, and satisfactory reduction of blood pressure in most cases.

A possible disadvantage is the effect of postural hypotension, particularly in cases liable to myocardial ischaemia. These effects may be largely prevented by careful adjustment of dosage at suitable intervals, since the drug has a cumulative effect.

In spite of our limited experience we feel that guanethidine is the drug of choice, and that its use represents a major advance in the treatment of severe hypertension.

Two of us (B.J.B. and J.E.P.T.) wish to thank the Director of Medical Services, Southern Rhodesia, for permission to publish.

Generous supplies of guanethidine (Ismelin) were made available for the purpose of this trial through the courtesy of Mr. J. H. G. Geer of Ciba Laboratories Ltd.

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TAALRUBRIEK

Die Taalkomitee van die Geneeskundige Skool van die Universiteit Stellenbosch stel voor om te gebruik:

1. Eng. *subscapular*: Afr. *subskapulêr*(e).
2. Eng. *interosseous*: Afr. *interosseus*(e).
3. Eng. *tuberosities*: Afr. *tuberositas*, mv. *tuberositasse*.
4. Eng. *malinger*: Afr. jou/hom *siek hou*, dus bv. Eng. 'He is malingering (again)': Afr. ‚Hy hou hom (weer) siek', of dit kan met 'n b.nw. weergegee word: hy is *skynsiek*.
   Malingering as s.nw.: *skynsiekheid*; *a malingerer*: *'n skynsieke*.
5. Eng. *malunion*: Afr. *wanhegting*. 'n *Non-union* (i.v.m. been) kan dan 'n *nie-hegting* wees, en 'a delayed union' ‚*'n vertraagde hegting*'.
6. Eng. *wheezing*: Afr. *fluit* (of ook: *gefluit*, van die bors).
7. Eng. *vesical calculus*: Afr. *blaassteen*.
8. Eng. *torpor*: Afr. *torpor*.
9. Eng. *pitting oedema*: Afr. *duik-edeem* (niemand hoef die koppelteken te gebruik as hy *duikedeem* vir die oog net so onmiddellik duidelik vind nie). Daarteenoor kan ons dan hê: *duikvrye edeem*.
10. Eng. *exteriorize*: Afr. *eksterioriseer* (ww., en dan as s.nw. *eksteriorisasie*). *Veruiterlik* en *uitlig*, *uitbring* ens. is ook oorweeg, maar *ver*uiterlik het 'n bygedagte van oppervlakkigheid en *uitlig* ens. is te algemeen.)
11. Eng. *a bypass*: Afr. *'n omleiding*. Wie dan wil of moet onderskei tussen Eng. *bypass* en *shunt*, sou in Afr. kon kies tussen *omleiding* en *aftak(king)*.
12. Eng. *intermittent claudication*: Afr. *kloudikasie*. Aangesien *kloudikasie* altyd ‚intermittent' is, vind die komitee dit oorbodig om vir dié woord in dié verband ook 'n Afr. ekwivalent te gee.

Korrespondensie

Baie dankie vir al die vriendelike briewe van alle provinsies en S.W.A. af. Die voorstelle, en plek-plek bedenkinge, wat daarin genoem word, word ernstig oorweeg en die besluite in dié verband sal later hier gepubliseer word.

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Unusual Cases

SACROCOCCYGEAL TERATOMA IN THE ADULT

A CASE REPORT

ARTHUR MEARS, M.B., B.CHIR. (CAMB. & ST. BART.), F.R.C.S. (EDIN.), *Department of Surgery, Groote Schuur Hospital, Cape Town*

Large sacrococcygeal teratomata are extremely uncommon in adults. The case presented describes a 15-lb. tumour in an intelligent adult female city-dweller who managed to adapt herself remarkably well to its presence.

CASE REPORT

A European female, aged 36 years, was admitted to the Conradie Hospital, Pinelands, Cape, on 2 August 1959 with a large tumour growing from the left buttock. She said that the tumour had been present from birth, and in childhood her parents had consulted 2 doctors about it. It had been decided to leave it alone. The only reason that had brought her to seek medical advice was that 2 days before admission she had felt ill, with shivering attacks, and had noticed an alteration in the appearance of the tumour.

Examination

On examination, the patient was an intelligent woman with a slight but well-proportioned physique.

Hanging from the left buttock in the region of the left ischiorectal fossa was an enormous tumour, as seen in the photographs, obviously coeval with the patient. The pedicle,

Figs. 1 - 5. Various views of the tumour.

which extended into the ischiorectal fossa, was in immediate proximity to the vagina and rectum and in fact distorted the anal orifice on the left side. The tumour was firm in consistency, and the skin over the major part had a *peau d'orange* appearance, with a dusky red colour.

The patient was pyrexial with a temperature of 102°F., and it was obvious that cellulitis of the skin over the tumour accounted for her general condition and the changes observed in the tumour. For this antibiotics were given. A straight X-ray picture of the pelvis and the tumour revealed no abnormality.

Operation

On 7 August 1959, under general anaesthesia, the tumour was removed. The patient was placed in the lithotomy position with the tumour resting on a separate table. The dissection was started through a circular incision of the pedicle. Many layers were encountered, one of which was recognizable as a stretched sheet of levator-ani muscle. Eventually the thin wall of an obvious cyst was identified. This cyst was found to fill the left ischiorectal fossa, in contact with the rectum and vagina, and in the final dissection was detached from the last piece of the sacrum and the coccyx. The operation took 3½ hours.

Specimen

This, essentially, was a large skin-covered cyst weighing 15 lb. in all. It contained free fluid and large amorphous masses of sebaceous material, as well as thousands of discrete 'sago-rice' grains of the same material. There was no evidence of other structures such as bones, teeth, hair, etc.

Pathological Report

'The cyst covering was composed of a dome of skin which was 8 inches in diameter. The centre of this dome of skin showed moderate excoriation. The cavity was uniloculated and about 8 inches in internal diameter with contents which were of a soft cheesy consistency and yellow-brown in colour. The cyst lining was generally smooth. The cyst wall was of white, leathery texture and about ¼ inch in thickness.

'The tissue subjacent to the skin consisted of subcutaneous loose connective tissue and layers of muscle into which were infiltrating bands of material resembling the contents of the cyst cavity.

'On microscopic examination, the contents of the cyst showed amorphous material resembling the contents of a sebaceous cyst.

'The lining consisted of avascular fibrous tissue with areas of chronic granulomatous inflammatory reaction and areas lined by cuboidal and ciliated columnar epithelium. One region showed an aggregate of muscle bundles.

'The subcutaneous tissue showed infiltrations of the material contained in the cyst with a chronic inflammatory reaction consisting of foam cells and giant cells.

'The histological features of the growth are those of a cystic teratoma.'

Postoperative Course

Convalescence was normal, and the wound healed by first intention. The function of the sphincter ani was undisturbed. It is interesting to note that it took some weeks for the patient to become accustomed to the absence of the tumour, especially on sitting down.

COMMENTS

Willis,[1] in his series of 82 cases, found the following distribution of teratomata: ovary, 50 cases; testis, 19 cases; and miscellaneous, 13 cases. The 13 miscellaneous cases were distributed at the following sites: epididymis, retroperitoneal space, anterior mediastinum, intrapericardial area, brain, and the presacral area.

Those in the presacral area are commonly referred to in the literature as sacrococcygeal teratomata, and by some authors as Middeldorpf tumours. The propriety of the latter terminology is disputed by others[4] who categorically state that Middeldorpf described neither a presacral teratoma nor any type of sacral neoplasm.

Sacrococcygeal teratomata are well-recognized entities and there is an abundance of case reports concerning them in the literature. One article[5] mentions 426 cases collected from the literature.

Briefly, these tumours are usually found in female infants and are generally obvious at birth. Many of these infants are either stillborn[2] or survive only a few days. The tumours may be solid, cystic, or mixed, and vary in size from that of a walnut to that of the baby's head. These tumours have a definite tendency to become malignant. This tendency has been expressed in different articles as 9%,[2] 17%[4] and 27%.[5] Further, there appears to be a definite association with the incidence of twins.[4,5] One paper[5] reports on a questionnaire sent to patients. Of 23 replies received, 12 confirmed the presence of twins in the family — an association of more than 50%.

Histologically, every kind of tissue, derived from the three germinal layers, has been reported in these tumours, and in some reports portions of limbs and organs are mentioned. The teratomatous cyst, such as the one described here, is a common type and may be confused with the inclusion dermoid cyst. Willis[1] discusses this matter thoroughly, and states that if the cyst wall is examined histologically, evidence of tissue of endodermal and mesodermal origin will be found, as in this case.

The origin of these tumours is a matter of great interest to the theorist, and the presacral area is a happy hunting ground for him on account of its embryological associations.

It is universally agreed that treatment by surgical removal offers excellent prospects of success. It is recommended that the coccyx[5] be removed with the tumour. The prognosis[5] is said to be better with the newborn than with the older child on account of the increased possibility of a malignant change.

DISCUSSION

The great interest of this case lies in the age of the patient and the enormous size of the tumour.

The vast majority of the case references in the literature are of infants and children. In one review of 72 cases,[2] 6 were between 4 and 18 years of age, and a further 6 between 20 and 60 years of age, but no details were given. In another report[6] the patient was aged 22 years, but the cyst involved was a very small one.

It requires little imagination to realize what a handicap a tumour of this size in this particular location could be in every-day life. Sitting down, the choosing of clothes to wear, going to the lavatory, keeping the tumour clean, etc., would be daily problems. Yet this patient had adapted herself so well that, except for an inability to take part in sport, she had led a normal life. She holds a responsible post as secretary, entailing a daily railway journey. She is married and had one pregnancy 12 years ago with a normal delivery of a healthy baby.

The patient, obviously an intelligent woman, states that the possibility of removal of the tumour never entered her head. She blames her parents, particularly her mother, who at no time ever discussed the tumour and its possible significance.

When she married, her husband did not mention the tumour or ask about it. As far as she knows, he is ignorant of its existence. The husband's attitude is odd, because even now he has never asked her for what

operation she has been to hospital. The midwife who confined her 12 years ago did not suggest that she should consult a doctor about the tumour.

In fact, there seems to have been a conspiracy of silence about this tumour by those closest to her. It would appear that she was brought up with the idea that the tumour would be an inevitable part of her existence for the rest of her life. As nobody had ever suggested otherwise, she had become resigned to its presence.

She is delighted to be rid of the tumour, particularly now that she can wear normal clothes and go swimming. She states that she is so adapted to her present state that she can hardly believe she ever had such a tumour.

SUMMARY

1. A case is reported of a 15-lb sacrococcygeal teratoma, dependent from the left buttock, in a 36-year-old European female. The tumour was successfully removed by operation.

2. The physical and psychical difficulties encountered and overcome by the patient while the tumour was present are discussed.

3. The pathology of sacrococcygeal teratomata is briefly discussed.

I wish to thank Drs. J. J. Hofmeyr, E. B. Lawton and H. S. M. van Jaarsveldt for their valuable assistance. Dr. Hofmeyr took the excellent photographs and Dr. R. Shapera prepared the pathological report. I must thank Dr. T. Jones, Medical Superintendent, Conradie Hospital, for access to the hospital records.

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INCIDENCE OF DIABETES MELLITUS IN BASUTOLAND : POSSIBLE NUTRITIONAL INFLUENCES

W. M. POLITZER, M.D. (PRAGUE), *South African Institute for Medical Research, Johannesburg*, and T. SCHNEIDER, M.D., F.R.C.P. (EDIN.), *Diabetic Clinic, General Hospital, Johannesburg*

A recent investigation into the prevalence of diabetes mellitus in the Butha Buthe district of Basutoland revealed an incidence of 0·23% among 3,000 individuals[1,1(a)] as against 1·7% in a White community of the USA[2] and 1·2% in Great Britain.[3]

The problem arises what possible factors are involved in the aetiology of diabetes mellitus.

Heredity

Diabetes mellitus is considered to be a hereditary disease[5,6] and susceptibility to it has been stated to be inherited *via* a simple recessive gene.[7] Joslin[8] showed the disease to be inherited in 60% of diabetic children under his care who lived longer than 20 years.

In the Bantu, family histories are notoriously difficult to obtain and it cannot be ascertained how far heredity plays a part in the incidence of diabetes mellitus.

Age

Age has always been considered relevant to the incidence of diabetes. In the USA one-quarter to one-third of the cases of diabetes have occurred before the age of 45 years.[9] This is borne out by the fact that among 69,088 male selectees for National Service in America between the ages of 18 and 37 years the incidence of diabetes was found to be 1·1%,[10] whereas the total incidence of 1·7% indicates that the percentage of cases in the upper age-group will be higher.

In our own cases[1,1(a)] the incidence of 0·23% was found in a population with an average age of 36·8 years, the average age of the diabetics being 50·6 years. In the under-40-year age-group the incidence of hyperglycaemia was only 0·05%, while in the above-40 age-group this had increased to 0·55%. Taking into consideration the shorter life expectancy among the South African Bantu,[4,11] a lower rate of diabetes would be expected. Our figures however, are so low that further explanation is necessary.

Obesity

The association of obesity with diabetes is frequently referred to in the literature, where obesity has been described as the commonest predisposing factor.[8,12-16] Thus, among 1,000 new cases of diabetes Joslin[8] found 77% to be obese. Long[17] suggests that 70 - 80% of diabetics were over-weight before the onset of the disease. Pomeranze[18] found a history of obesity before the clinical presence of diabetes in 83% of 1,140 diabetic patients. Furthermore, the susceptibility to diabetes has been found to increase with the degree of overweight. Dublin[19] showed that the higher the percentage of overweight in men the higher the death rate from diabetes. While this was marked in the younger age-group the disproportion was even higher in the over-45 age-group (see Table I). Studies among women showed the same tendency.[20,20(a)]

TABLE I. OVERWEIGHT AND DIABETES MORTALITY

Above average weight	*Diabetes mortality above normal*	
	Under 45 years	*Over 45 years*
5 - 14%	1·5 times	2 times
15 - 24%	3·2 times	4 times
25% or more	8·5 times	10 times

The body build of the group investigated by us was in the main tall and thin; a few, however, were obese, particularly among the older females. Of the 7 cases showing hyperglycaemia only 1 was obese.

Calorie Intake

The association of obesity with excessive intake of calories is well known. Fleisch[21] has shown a direct relationship between the standard of living of the population in the different Swiss cantons and the incidence of diabetes. The low-calorie diets of the war years would appear to have had a remarkable effect in reducing the incidence and mortality of diabetes mellitus.[22-25] On the other hand it is a well-known fact that after a prolonged fast carbohydrate ingestion may raise the blood sugar high enough to cause glycosuria. The possibility of one or more of our cases having been affected in this way cannot be excluded. Himsworth[26,27] has con-

sidered a high proportion of fat in the diet rather than that of carbohydrate as favouring the development of diabetes, but a low-fat diet *per se* has not been found to influence its incidence. For example, diabetes occurs frequently among the Ceylonese, who consume a low fat diet, although the late manifestations of the disease would appear to be less than in people living on a normal mixed diet.[28] A recent communication, in fact, suggests that a low-fat diet—the fat content being limited to 20 g. per day—favours the regression of retinopathy in established cases.[29]

The population investigated by us was on a low-calorie, low-fat diet. The diet consisted of maize and kaffir corn (in the form of beer and porridge), and vegetables such as wild spinach, pumpkin, peas and beans as the main items. Meat and eggs were rarely partaken of, and milk was obtainable in small quantities during the winter months only.

Munoz and Anderson[30] recorded the calorie adequacy in Basutoland as being 87% of average normal intake.

Vitamins and Endocrines

It is to be expected that in the type of diet found among the Basuto, deficiency of vitamins will occur. Clinical signs of riboflavin deficiency in the population investigated are widespread.[31] Ershoff[32] has shown that deficiency of riboflavin markedly increases the insulin sensitivity of the rat, so much so that in some cases there occurred a pronounced and frequently fatal hypoglycaemia. It would appear, therefore, that this deficiency may be one of the factors in the low incidence of diabetes among the Basuto.

Among other factors to be considered in our series is endocrine imbalance. Munoz and Anderson[30] undertook a goitre survey in the lowlands of Basutoland and found an average incidence of 32%. A committee of the Department of Nutrition of the Union of South Africa in 1955[33] found that the whole of the Drakensberg area including the whole of Basutoland could be regarded as an endemic goitre area.

The combination of hypothyroidism with diabetes is not at all common. Joslin[8] reports only 11 cases among 32,148 true diabetics, and while endemic goitre may not be associated with marked hypothyroidism it can be expected that the incidence of diabetes in an endemic goitre area may be lower than in iodine-sufficient regions. The association of hyperthyroidism with diabetes is well known but hyperthyroidism is rare in the Bantu[34] and was definitely not a factor in the cases described by us.

The relationship of starvation and undernutrition to a lowering of the activity of the pituitary gland, particularly of its anterior lobe, is well known.[35] It has been stated that undernutrition has the same effect as hypophysectomy.[35] Soskin and Levine[36] reported that depancreatized dogs on deficient diet, i.e., on protein only, showed the same amelioration of their diabetes as was demonstrated in dogs that had their pituitary removed. Young's experiments[37] showed that injection of crude or purified growth hormone from the anterior pituitary produced a diabetic state. However, this induced diabetes was considerably improved by fasting, and even complete recovery took place provided that the β-cells of the pancreas had not yet been completely destroyed.[37] It is possible therefore, that the low-calorie diet of the Basuto may have an inhibitory effect on the anterior pituitary, which may be a further factor in the low incidence of diabetes.

Similarly, it is possible that hypofunction of the suprarenal gland may play a part. Thus Politzer and Tucker[39] have found a significant paucity of 17-ketogenic hormone in the Bantu male. This finding, as well as the related one of small suprarenal glands in West Africans,[40] indicates a deficient production of glucogenic hormone in the African. If the low hormone production leads to decreased gluconeogenesis it may be postulated that a lower incidence of glycosuria and hyperglycaemia will be initiated in these people. However, it must be acknowledged that there is little to point to adrenocortical hypersecretion as a cause of clinical diabetes apart from its occurrence secondarily to conditions such as Cushing's syndrome and phaeochromocytoma.

Iron Metabolism

In contradistinction to the factors influencing the low incidence of diabetes is the occurrence of siderosis in certain parts of Africa.[41-43] Heavy deposits of iron pigment in the viscera have been described by several authors.[41-43] The iron pigment may be deposited in the reticulo-endothelial cells of the liver, in which case the condition may be asymptomatic, or it may lead to parenchymal deposition, in which case symptomatic haemochromatosis may ensue. Dodu[44] in a series of 44 diabetics in Ghana found heavy iron-pigment deposits in the bone-marrow in 7 cases. In 5 of 6 cases in which liver biopsy was performed haemochromatosis was diagnosed. The Basuto like many other Bantu use iron cooking utensils and beer-making receptacles. It is natural to assume that they too are consuming a considerable quantity of iron. The possibility, therefore, emerges that some cases of diabetes may find their origin in haemochromatosis.

CONCLUSION

A number of factors bearing on the incidence of diabetes mellitus have been discussed.

The influence of heredity and age is, of course, predominant, but that of diet and nutrition has been shown to be of great importance. In various countries the reduction of calories during the war years with its associated lowering in diabetic incidence suggests that reduction in calories may cause the hereditary tendency to diabetes to remain latent. It is difficult to estimate to what extent the other factors mentioned may have a bearing on the low incidence of the disease in the Basuto, but hormonal factors secondary to the low-calorie diet cannot be discounted. Likewise, although there is no evidence that diabetic incidence is markedly reduced in countries where fat intake is small, recent studies in fat metabolism and its relationship to atherosclerosis[45-48] and diabetes mellitus[49] (including the late manifestations of the disease[50]) would point to the necessity for evaluation of a low-calorie, low-fat diet as a preventive measure in those hereditarily predisposed to this disease. In this connection we would particularly refer to those in whom the hereditary trait is found in both sides of the family.

Finally, we would suggest that the low-calorie, low-fat, high-carbohydrate diet taken by the Basuto and other African tribes may be the main factor in their being preserved from the discomfort and progressive development of diabetes mellitus and its late manifestations.

SUMMARY

1. The incidence of diabetes mellitus was found to be 0·23% among 3,000 individuals in the Butha Buthe district of Basutoland.

2. While hereditary influences and age are of importance in the incidence of the disease, nutritional factors must be considered.

3. The problem is discussed from the point of view of obesity, caloric and fat intake, vitamin deficiency, and endocrine imbalance.

4. The rôle of dietary iron is commented on.

5. The lowered incidence of diabetes mellitus in various countries during the war years is stressed as suggesting that reduction in calories and fats may cause the hereditary tendency to diabetes to remain latent.

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QUESTIONS ANSWERED : VRAE BEANTWOORD

CONSTIPATION

Q—*What is the best approach to the problem of chronic constipation,* (a) *in general, and* (b) *in patients who are subject to the spastic-colon syndrome?*

A—Many people think that they are constipated if they do not have a motion every day. The bowel habits of people may vary enormously, some have 1 motion a day, others have 2 - 3 a day, while other people have a motion only every 2 - 4 days. As long as a person feels comfortable, he need not worry about omitting a day without having a motion. The idea which was current earlier in this century, that the effect of constipation was the production of 'auto-intoxication' bringing in its train all sorts of ills, from rheumatism to headaches, from tiredness to nervousness, is not now held to be valid. It is however quite true that in some individuals chronic constipation may lead to uncomfortable sensations. But very frequently patients will resort to purgatives because they are under the impression that if they don't 'clean their bowels' every single day, then they are not normal and may become liable to all sorts of disease.

The first thing to do in cases of chronic constipation — excluding of course those cases which are caused by organic disease — is to stop all purgatives. The patient may not have a daily motion, but after an interval of time he may get into a rhythm of his own with which he is quite comfortable.

When chronic constipation is associated with the spastic-colon syndrome — a common association — then the problem is complicated by the fact that such people often have considerable abdominal pain and flatulence. If they are constipated for any length of time, they feel distended and uncomfortable, and if they take even a mild purgative, then their irritable colon reacts with a bout of diarrhoea, or they develop abdominal pain. For such cases it is advisable to stop, for a time, all purgatives and roughage and to give enemas (every alternate day) for a few weeks. At the same time, the administration of phenobarbitone and belladonna and the application of heat to the abdomen are very useful and are a great comfort. After a few weeks the enemas are given at less frequent intervals, and then discontinued, by which time the patient may be fortunate enough to have normal motions on his own.

What about purgatives? They should be reserved for people who are too old to be trained into new bowel habits, for those who are feeble and ill, and for those few desperate people who will not respond after a long trial of re-education. With regard to the actual drugs employed, it is found that liquid paraffin may be given to most patients, although it will probably be found to have little effect on obstinate cases of constipation. The bulk-producing substances like agar-agar may suit some patients, but are often inadequate in cases of severe constipation and may produce pain in those suffering from the spastic-colon syndrome. In the few cases where one is forced to resort to purgatives, the anthracene derivatives, such as cascara and phenolphthalein, are less irritating in patients with spastic-colon syndrome, than are magnesium sulphate and 'fruit salts'. In some patients, glycerine or bisacodyl suppositories may be adequate and well tolerated.

A diet containing a large amount of cellulose — a high roughage diet — may be disappointing, and in some people it tends to produce flatulence and abdominal discomfort.

Every normal person should, however, include in his diet a reasonable amount of fruit, vegetables, and salad. A glass or two of some milk or yoghurt taken on an empty stomach might be useful. Vitamin-B compound and yeast preparations are often prescribed, but their efficacy in obstinate cases is doubtful. The patient should be told to go to stool at a regular time every day — preferably after breakfast — but he should avoid pressing. Re-education of the patient is still the most valuable aid.

Readers are invited to submit questions for this column to the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town.

IN MEMORIAM

ISRAEL MARIN, M.B., B.CH., B.A.O. (DUBL.)

Dr. W. P. Steenkamp, of Cape Town, writes:

The sudden death of Dr. I. Marin was a great shock, particularly to me, since we had been friends for 30 years.

After qualifying in Dublin, Dr. Marin settled in Brandvlei, Cape. It may seem a mystery why he, a city-bred man, should have picked this out-of-the-way village in the North West Cape, but it can be explained, however, by the innate love of the soil and the wide open spaces which always appealed to the philosophical side of Dr. Marin's nature. Dr. Marin later moved to Calvinia, on being appointed district surgeon, and he practised there until recently, when he decided to take things more easily and devote himself to doing *locum tenens* in the Cape Peninsula.

Dr. Marin

The North West Cape, when Dr. Marin settled there, was a drought-stricken area with poor communications and roads and without hospital facilities or antibiotics; it was indeed a testing ground for any medical practitioner. However, Dr. Marin overcame these difficulties remarkably well. He was probably the best medical 'allrounder' I have ever met in many years of general and consultant practice. He received his postgraduate training in a hard school where he had to make, and act on, his own decisions. Not only was he a good general-practitioner surgeon capable of handling all emergencies efficiently, but he was also an able obstetrician who very seldom resorted to that drastic panacea for the majority of labour complications, namely, Caesarean section.

One of the tragedies of his life (tuberculosis in a near member of his family) led to his great interest in this disease, and he ultimately became an expert on tuberculosis. He also became interested in anaesthetics and could hold his own in this specialized field.

His interests in sport were very wide. Among other things, he contacted and persuaded the late Sir Malcolm Campbell to try out his Bluebird I at Verneukpan. Dr. Marin was also a keen golfer.

At Calvinia he worked tirelessly until a hospital, the first in the North West Cape, had been built, and became its first medical superintendent. He also excelled as a farmer and, although his activities in this field were on a small scale, he did much by his experiments to educate his neighbours in the importance of different grasses under irrigation. Being an experimental farmer, however, who farmed for interest only (as quite a number of colleagues I know), he never gained financially but, on the contrary, had to work hard at his profession of medicine to support his hobby. As a result Dr. Marin never accumulated money, but he also did not suffer a coronary thrombosis — the inevitable reward of the one-track medical man.

Dr. Marin was a keen freemason and, thanks to his efforts, freemasonry was kept alive at Calvinia and the Rebunie Lodge, which was built in the time of the depression by a mere handful of brethren at Calvinia, today stands as a monument to the man who probably held the unique record of having been master of his lodge for 5 different years.

Dr. Marin was not only a well-read philosopher, which sustained him during a life full of hardship, trials and tribulations, but he also had a keen sense of humour. His favourite story was of a suspected case of typhoid he was called in to see. Being an honest man, which unfortunately counts against a successful bedside manner, he explained to the family that to settle the diagnosis he would call early the next morning to collect a sample of blood in time to be sent by post to Cape Town for a Widal test. He would then be notified of the result telegraphically within 24 hours. Early the next morning however a little girl arrived quite out of breath to announce 'Ma het laat weet dat dokter nie meer Annatjie se bloed hoef te kom neem nie want Tant Sannie was nou daar en sy sê dit is maagkoors wat Annatjie het'.

Dr. Marin was a reliable friend and loyal colleague, with malice towards none, not even those who had let him down, and it was a privilege to have known him.

HENDRIK STRYDOM SWART, M.B., CH.B. (PRET.)

Dr. L. Jansen, van Rustenburg, Transvaal, skryf:

Dr. Hendrik Strydom Swart is gebore op 1 Januarie 1932. Hy het aan die einde van 1956 sy graad in die medisyne aan die Universiteit van Pretoria behaal. Hy het as huisdokter diens gedoen aan die Boksburg-Benoni-Hospitaal en daarna op Nigel en Duiwelskloof gepraktiseer. In Februarie 1959 het hy hom op Boshoek, in die distrik van Rustenburg, gevestig.

Dr. Swart

Op 12 September 1960 is hy op slag gedood in 'n motorongeluk net buite Rustenburg terwyl hy op pad was om 'n operasie te doen. Hy is aan die einde van 1956 getroud en laat sy vrou en een seuntjie na.

Die hele gemeenskap van Rustenburg is diep geskok oor sy vroegtydige afsterwe.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. M. Jordaan, M.D., Ph.D., borschirurg, van Kaapstad, het na sy onlangse siekte weer sy praktyk hervat te Mediese Sentrum.

* * *

South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-Group will be held on Tuesday 13 December at 8.15 p.m. in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape. Dr. David McKenzie will speak on 'Some aspects of liver disease in childhood'. Visitors are welcome to attend this meeting.

* * *

Dr. V. D. Bokkenheuser, of the South African Institute for Medical Research, Johannesburg, has been appointed to a Research Fellowship at the Children's Hospital, Buffalo, USA. Dr. Bokkenheuser will spend a period of 1 year with Dr. E. Neter studying the receptor sensitivity of erythrocytes under diverse pathological conditions.

Dr. W. P. U. Jackson, of Cape Town, has been granted a Wellcome Trust Travelling Fellowship for June - July 1961 to visit London and the Scandinavian countries while he is in Europe next year.

* * *

British Council Specialist Courses for 1961. Among the courses to be organized by the British Council are: (1) Anaesthesia, 19 February - 4 March, London and Oxford. (2) Use and evaluation of drugs in medicine, 30 May - 13 June, Macclesfield, London, Oxford. (3) Plastic surgery, June, East Grinstead. Further details of these courses will be published when they become available.

* * *

Dr. H. Hamersma, M.B., Ch.B. (Pret.), M.D. (Amsterdam), oor, neus en keelarts, het op 1 November verhuis na nuwe spreekkamers te Van Riebeeck Mediese Gebou 622, Pretoria. Die telefoonnommer bly onveranderd — 3-3444.

Society for Clinical and Experimental Hypnosis (Southern Section). This Society will present a tape-recording evening on Tuesday 13 December at 8.15 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. The programme will include 'Use of self-chosen pressure points for induction and deepening of the hypnotic state' by Dr. David B. Cheek, M.D., of the Department of Obstetrics and Gynecology, Mount Zion and Children's Hospital, San Francisco, California; 'Theory and techniques of induction' a discussion by a panel including Bernard Raginsky, President of the International Society for Clinical and Experimental Hypnosis and Dr. W. Schaeffer, psychiatrist; and 'Specialized hypno-analysis technique (whirling stars)' will also be discussed. All interested are invited to attend this meeting.

* * *

Mr. Herman Klein, M.Ch. Orth. (L'pool), F.R.C.S. (Edin.) has commenced practice as an orthopaedic surgeon at 310 Lister Building, Jeppe Street, Johannesburg. Telephones: Rooms 23-9625, residence 44-0150.

Dr. Herman Klein, M.Ch. Orth. (L'pool), F.R.C.S. (Edin.), het nou begin praktiseer as 'n ortopediese chirurg te Listergebou 310, Jeppestraat, Johannesburg. Telefone: Spreekkamer 23-9625, woning 44-0150.

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering. Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie van adres verander.

* * *

Mr. J. N. Bear, M.B., B.Ch., F.R.C.S. (Edin.), formerly first assistant in the Department of Orthopaedic Surgery at the Addington Hospital, Durban, and registrar in orthopaedics at the Middlesex Hospital, London, has commenced practice as an orthopaedic surgeon in partnership with Mr. Cyril Kaplan, M.Ch.Orth., F.R.C.S. (Eng.), at 70 - 74 Trust Building, Gardiner Street, Durban. Telephones: Rooms 28272, residence 20793.

Dr. J. N. Bear, M.B., B.Ch., F.R.C.S. (Edin), voorheen eerste assistent in die Departement van Ortopediese Chirurgie by die Addington Hospitaal, Durban, en registrateur in die ortopedie te Middlesex Hospitaal, Londen, het nou begin praktiseer as 'n ortopediese chirurg in vennootskap met dr. Cyril Kaplan, M.Ch.Orth., F.R.C.S. (Eng.), te Trust-gebou 70 - 74, Gardinerstraat, Durban. Telefone: Spreekkamer 28272, woning 20793.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during October 1960 are gratefully acknowledged:
Met dank word die volgende skenkings gedurende die maand Oktober 1960 erken:

Votive Cards in Memory of : Geloftekaarte ter Nagedagtenis aan:
Mrs. Nel *by* Dr. A. H. Baxter; Dr. S. A. Lange *by* Mr. L. R. Holwill.

Total Received from Votive Cards: £2 2s. 0d.
Totaal Ontvang van Geloftekaarte:

Services Rendered to : Dienste Gelewer aan:
Dr. E. Schultz *by* Drs. S. Kay, J. Gluckman and L. Varejes.
Wife of Mr. R. P. Hewitson, F.R.C.S. *by* Dr. H. G. Rees.
Dr. W. F. Skaife *by* Dr. G. W. Moggridge, Dr. N. K. Pein and Dr. R. J. H. McMahon.
Dogter van dr. A. M. le Roux *deur* Drs. Grundlingh, Viljoen en Johnson.
Late Mr. E. Jenkin *by* Dr. F. Wood, Dr. H. Brown, Dr. A. A. Brown, Dr. P. le Roux and Dr. A. McCallum.
Mrs. I. A. Lurie *by* Dr. W. H. D. Trubshaw, Dr. J. N. Smith, Dr. F. P. Reid and Dr. J. N. Jacobson.
Dr. E. L. Dirmeik *by* Drs. D. W. Burton, H. H. Bloch and E. W. C. Emms.
Mrs. Dirmeik *by* Dr. A. W. Sichel.
Mr. S. Dirmeik *by* Dr. E. W. C. Emms.

Total Received from Services Rendered: £72 17s. 0d.
Totaal Ontvang van Dienste Gelewer:

Donations : Skenkings:	£	s.	d.
Dr. H. H. Broodryk, Dr. R. T. Hayes	1	1	0
Cape Western Branch (M.A.S.A.) — collection box	2	14	11
Drs. Harries, Kuschke and Hofmeyr	1	1	0
Dr. L. Kirshon	1	1	0
Drs. Segal, Karstaedt and Geffen	3	3	0
Total Donations : Totaal Donasies	£9	0	11

Grand Total : Groot Totaal £83 19s. 11d.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

VERACTIL SOLUTION

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of a parenteral presentation of Veractil brand methotrimeprazine, a compound of particular value in the management of anxiety and agitation and in the relief of severe pain.

Hitherto Veractil has been available as tablets of 5 mg., 25 mg., and 100 mg., and the injectable solution will be found useful for the treatment of the more severe cases, or when patients are unable to take tablets.

Veractil injection is available as a 2·5% w/v solution of methotrimeprazine hydrochloride in boxes of 10 1 ml.-ampoules.

Further information may be obtained from Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

STELABID

SKF Laboratories (Pty.) Ltd., announce the introduction of Stelabid, and supply the following information:

Stelabid is a long-acting preparation which combats cause and effect in peptic ulcers and other stress and stomach disorders.

Stelabid is indicated in peptic ulcers and a wide range of gastro-intestinal disorders, especially where anxiety, tension, and other nervous or emotional factors are thought to be responsible, either wholly or in part, for the condition.

Anxiety, frustration and similar manifestations of nervous or emotional stress are known to play a vital part in the aetiology of peptic ulcer and other gastro-intestinal disorders. Effective treatment of these conditions, therefore, should do more than provide transient relief of distressing local symptoms — it should also act decisively at the source of the trouble. Stelabid places in the hands of the practitioner the means of implementing this essential dual approach to therapy.

Actions. Stelabid contains 'stelazine' (trifluoperazine), now established as one of the most effective of the tranquillizing derivatives, and 'tyrimide' (isopropamide iodide), a powerful anticholinergic drug with a day-long duration of action.

Stelabid relieves anxiety and tension in the patient, effectively reduces gastric secretion, inhibits hypermotility and spasm, and prevents the nausea and vomiting that frequently accompany these disorders. Thus Stelabid gives complete control of both cause and effect in digestive disorders arising from, or aggravated by, stress.

Contra-indications and side-effects. Stelabid should not be prescribed for patients with glaucoma, pyloric obstruction of organic origin, or prostatic hypertrophy.

Care should be taken in treating patients with cardiovascular

impairment, patients receiving opiates, sedatives or other depressant drugs and in treating children under 12 years of age.

Minor side-effects, such as dry mouth, blurred vision, restlessness or insomnia, constipation and, more rarely, urinary hesitancy may occasionally be encountered. Extrapyramidal effects are not to be expected, as the dosage of the Stelazine constituent is low.

Dosage. One tablet *b.d.* or *t.i.d.*, according to the severity of the condition.

Stelabid tablets are supplied in containers of 30.

Further information may be obtained from SKF Laboratories (Pty.) Ltd., P.O. Box 38, Isando, Transvaal.

BOOK REVIEWS : BOEKBESPREKINGS

DIAGNOSTIESE RÖNTGENOLOGIE

Einführung in die Röntgendiagnostik. Von Prof. Dr. U. Cocchi und Priv.-Doz. Dr. P. Thurn. viii + 339 Seiten. 419 Abbildungen in 547 Einzeldarstellungen. DM 49.50. Stuttgart: Georg Thieme Verlag. 1959.

Hierdie boek gee nie voor om iets meer te wees nie as 'n inleiding tot die diagnostiese röntgenologie. Dit is dus geen naslaanwerk nie. Soos die skrywers dit self stel, is dit bedoel vir studente in die kliniese jare wat op die hoogte moet wees van die tegniek, wese, en grense van diagnostiese röntgenologie, en vir praktiserende geneeshere wat belangstel in hierdie hulpmiddel. Hiermee slaag Professor Cocchi, bekende leermeester en navorser, uitstekend in sy doel.

Dit is interessant dat daar in die geskiedkundige oorsig 'n herdruk is van Röntgen se oorspronklike publikasie 'Oor 'n nuwe soort van strale' in 1895. Dat Röntgen destyds reeds soveel fisiese eienskappe van die onbekende strale kon bepaal, is nog steeds 'n bron van verwondering.

Na 'n duidelike bespreking van die tegniese en fisiese beginsels, apparate en inrigting van die röntgenafdeling, volg daar die gewone hoofstukke oor botte, gewrigte en inwendige organe. Sommige illustrasies, veral van die longe, kon beter gewees het. In ander opsigte is dit een van die beste werke in sy soort wat ek nog teëgekom het. A.D.K.

BREAST CANCER

Breast Cancer. The Second Biennial Louisiana Cancer Conference. New Orleans, January 22-23, 1958. Edited by Albert Segaloff, M.D. Pp. 257. 43 Figures. South African price. £2 2s. 6d. Local agents: P. B. Mayer, P.O. Box 713, Cape Town, and Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg. St. Louis: The C. V. Mosby Company. 1958.

This monograph gives an excellent summary of the various points of view currently prevalent in the United States on the most intriguing problem of breast cancer. The various papers presented come under the sections of Basic biology, Definitive treatment, and Hormonal therapy. In the section on basic biology Emerson Day presents a full epidemiological survey and, amongst many interesting observations, notes the much more frequent prevalence of the disease in the West than in the East. No reasons are advanced for this and it presents a fruitful field for future investigation. The various causative factors that might initiate breast cancer are adequately discussed; so is the cytology of breast secretions. Emphasis is laid on the extreme variability in behaviour of breast growths. Besides differences in morbid anatomy and histology there are variations of heredity, age, hormonal type, menstruation, pregnancy, and the menopause in the host.

In the section on definitive treatment all the known methods of treatment have their various advocates, from the extended radical operation of Urban, through Haagensen's triple biopsy before radical mastectomy, to Garlands' championing of the MacWhirter technique of simple mastectomy followed by radical radiotherapy. It is interesting to note that Haagensen is narrowing his criteria for operability most rigidly, advocating operation only for the cases he feels can be cured, and condemning surgery in other cases as harmful and contra-indicated. Garland, in an excellent paper, very adequately answers Ackerman's criticism of the MacWhirter technique. Escher gives a short review of the various cytotoxic and cytostatic agents used in advanced disease.

In the section on hormonal therapy detailed descriptions are given of the value of the oestrogens, androgens, cortical steroids, and endocrine ablative measures employed; and the great variability of the results obtained in different clinics add to the uncertainty experienced in deciding which patient will benefit from these measures.

The impressions one is left with after reading the views expressed at this conference is that the problem of breast cancer remains a most formidable one calling for renewed combined efforts for its solution. I.M.S.

FLUID BALANCE

Essentials of Fluid Balance. 2nd edition. By D. A. K. Black, M.D., F.R.C.P. Pp. xii + 135. 6 figures. English price 20s. Oxford: Blackwell Scientific Publications, Ltd. 1960.

The second edition of Professor Black's monograph incorporates minor changes to bring it up to date. The text is easy to read despite the large number of references for so small a book, and the figures are good. The style is clear and sometimes witty. The first 5 chapters deal with the general properties of body-fluid; water, sodium, potassium, and anions and acid-base balance; and in the 6th the author submits a helpful approach to treatment. Some useful additional information is appended. In the bibliography Professor Black draws attention to recent review articles, setting an example which other authors might profitably follow in books of this type.

The author has two aims, viz. to keep the 'book as short as possible, consistent with clarity', and to 'make the presentation somewhat general', and in these he has succeeded admirably. There must be few practitioners today who do not need to renew their acquaintance with the principles of fluid balance and to keep abreast of recent advances; and no better way can be recommended than to read this book. E.B.A.

FOETAL OXYGEN

Oxygen Supply to the Human Foetus. A symposium organized jointly by The Council for International Organizations of Medical Sciences. Edited by James Walker and Alec C. Turnbull. Pp. xii + 313. Figures. 47s. 6d. Oxford: Blackwell Scientific Publications Ltd. 1959.

Safer abdominal delivery has solved many of the problems related to the mechanics of childbirth. The solution to present-day obstetrical problems seems to be bound up with a better knowledge of the anatomy and physiology of the placenta. The report of the symposium held in Princeton in December 1957 reflects the new attitude of obstetricians, with special reference to research on oxygen supply to the foetus.

The 22 participants included such famous research workers as Walker (Dundee), Browne (London), Bartels (Germany), Metcalfe (Massachusetts), Minkowski (Paris), Prystowsky (Florida), MacKinney (Carolina), and Rooth (Lund).

The subject matter includes the vascular anatomy of the placenta, physiology of oxygen transport in the adult and the foetus, studies of uteroplacental blood flow and oxygen consumption, clinical studies of cord blood-oxygen levels, laboratory techniques, and asphyxia and clinical foetal distress. The discussions of the papers are published in full and add interest.

This research is still in its infancy, as shown by the fact that the drop in oxygen saturation associated with increase in gestation originally reported by Walker, is not confirmed by Rooth, Minkowski, or Prystowsky. The discussions of this aspect by these workers themselves is the most interesting part of the book.

The obstetrician interested in foetal distress and postmaturity will find this a most interesting book. J.N.dV.

HYPNOTISM

Harley Street Hypnotist. A doctor's story as told by Alan Mitchell. Pp. 248. 15s. 6d. London, Toronto, Wellington, Sydney: George G. Harrap & Co. Ltd. 1959.

Although a summary of the contents of this book by the publishers suggest that it 'will appeal to medical and dental professions for its authentic and accurate information', it is felt that a work like this is of limited value to the profession. On the contrary, it may certainly appeal to the lay public, for it is written in fictional style but based on authentic fact. The first half of the book comprises a series of lectures on hypnosis which were actually delivered in London in 1957. A light-hearted fictional style pervades these lectures, each of which constitutes a chapter of the book. It holds very little of stimulating academic interest to the professional man.

The second half of the book relates some run-of-the-mill stories which abound in many doctors' case records, and which may again find possible favour in a lay journal.

This book reflects, in my opinion to disadvantage, on the common assumption by the public that it is easy to attain proficiency in the medical use of hypnosis; such an impression is in contradiction to the attempts of various medical, dental and psychological bodies to establish hypnotherapy firmly on a scientific basis, which necessitates the completion of extensive training courses in the subject of hypnosis and the allied sciences.

M.V.S.

HYPNOSIS

Hypnosis, Fact and Fiction. By F. L. Marcuse. Pp. 224. 3s. 6d. Middlesex: Penguin Books. 1959.

This is an extremely easy to read Pelican. The style is bright and witty. Every aspect of what has now become an enormous subject is discussed in terms of facts and fallacies. Hypnotic techniques, hypnotists, patients, therapy, and the dangers of hypnosis, are all described and analysed.

The writer says, 'The book is intended both for general reading and for supplementary reading for courses in introductory psychology, psychopathology, personality and the like'. I have no hesitation in recommending the book for the lay general reader, though I wonder what impression Marcuse's own stagy demonstration of hypnosis (p. 181) will make on the lay mind, in view of the deprecatory attitude the author laudably adopts towards stage hypnosis. While the writer says that his material 'comes from books, professional journals, magazines, lectures, newspaper articles and personal experience', no reference is made in the text to any of the serious present-day writers. This is a lamentable omission for the student who may wish to examine the source of the material more fully.

Finally, it is commendable that Marcuse maintains a sane perspective in viewing hypnosis as an aid in therapy and not as an extravagant panacea for all ills. B.W.L.

ANAESTHESIA

A Practice of Anaesthesia. By W. D. Wylie, M.B. (Cantab.), M.R.C.P., F.F.A.R.C.S. and H. C. Churchill-Davidson, M.A., M.D. (Cantab.), F.F.A.R.C.S. Pp. xv + 1056. 246 figures. 105s. net. London: Lloyd-Luke (Medical Books) Ltd. 1960.

In 1916 Paluel Flagg could justifiably insist that the practice of anaesthesia was an art alone. Although rabid disciples of science may object, modern anaesthesia remains an art, but an art based, not as in Flagg's day upon anatomy and empiricism, but ever more firmly upon a deepening understanding of physiology and pharmacology.

The present volume is a surprisingly detailed description of the very considerable foundation of anatomy, physiology, pharmacology and pathology upon which the St. Thomas's school of anaesthetic artists base their techniques. It is a text highly to be recommended on several grounds. Firstly, it emphasizes the importance of solid foundations and adequate structural framework rather than the minutiae of decorative plinths and cornices which so many unthinkingly believe to be the hallmarks of true worth. Secondly, it covers almost the whole field of clinical anaesthesia without being either too superficial or too detailed, an almost impossible task for such a rapidly expanding speciality. Thirdly, it is authoritative without being dogmatic, a virtue possessed by too few books and articles in the present world of medical literature.

No doubt there will be many who will disagree with some of the facts and opinions set forth by the authors and their contributors and, as in all new books, there are minor factual and printing errors awaiting discovery by the patient critic, but the volume is intended not as an ultimate source of authority but as an exposition of a particular philosophy of anaesthesia and the application of that philosophy.

Impeccably printed on glossy paper with ample line and photographic illustrations, the value of the book is further enhanced by very adequate lists of references arranged by chapter, and by a *vade mecum* and index. At 5 guineas the book is in fact extraordinarily cheap and should be in the possession of all occasional as well as all specialist anaesthetists.

C.S.J.

HELMINTHOLOGY

Medical Helminthology. By J. M. Watson, D.Sc. (Lond.), A.R.C.S. Pp. 500. 62 pp. of illustrations incorporating over 600 individual drawings. 84s. + 2s. 9d. postage. London: Baillière, Tindall & Cox Ltd. 1960.

Dr. Watson has attempted to include in one reasonably sized volume all the basic facts regarding the helminths parasitic to man, and one feels that he has done this in a most creditable manner. The result is a book which should prove of interest and value not only to undergraduate and post-graduate students but also to practising clinicians, pathologists, and public-health authorities.

The work is divided into 3 convenient parts. In Part I there is a general introduction to helminths and helminthic infections, and I found the historical review particularly entertaining. Part II forms the bulk of the book and comprises a systematic account of the helminths parasitic to man. Finally, there is a clinical section in which chapters are devoted to the diagnosis, treatment, and prevention of helminth infections. There is a valuable appendix in which helminths found in common laboratory animals are listed in a convenient table.

Helminthology is a subject which is currently receiving much attention from public-health authorities and research workers all over the world. The author has therefore wisely included a special chapter covering the most important recent advances in this field, including those which first appeared in print when the rest of his book had already reached proof-reading stage.

The book is easy to handle, the print is large and clear, and I am impressed by the value of the inclusion of natural-size silhouettes of the more important worms. M.I.S.

THE ARTERIAL WALL

The Arterial Wall. Edited by Albert I. Lansing, A.B., Ph.D. Sponsored by The Gerontological Society, Inc. Pp. 260 + ix. 17 pages of illustrations. 60s. + 2s. 6d. postage. London: Baillière, Tindall and Cox Ltd. 1959.

The publication of this monograph was made possible through the generosity of a layman in a grant to the Gerontological Society.

The knowledge to date on the structure, physiology and biochemical processes of the arterial wall is authoritatively reviewed by authors who have made original contributions to their subject. Each chapter is clearly and comprehensively presented and includes a generous bibliography. The chapters presented discuss: the vasa vasorum, the vascular endothelium, arterial muscle, collagen and ground substance, elastic tissue, mucopolysaccharides, enzymes, lipid metabolism of connective tissue, and the metabolism of the arterial wall. Only scant reference is made to pathological processes. One of the aims of the book in providing a summary of our knowledge was to stress what we don't know, and this it certainly achieves.

This review will prove interesting to the informed practitioner and most valuable to research workers in this and related fields.

An analogy is often drawn between the circulatory system and a plumbing system. This monograph should finally end any further use of such a simple concept. B.B.-S.

MANUAL OF SURGERY

Rose and Carless Manual of Surgery. 19th edition. Consulting Editor: Cecil Wakeley, Bt., *K.B.E.*, *C.B.*, LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. Pp. xv + 1389. Illustrated (12 colour plates). 84s. London: Baillière, Tindall and Cox Ltd. 1960.

The 19th edition of this volume published this year carries on a long tradition. The first edition appeared in May 1898 and since that time the manual has become widely known.

It is of particular value as a reference work for students and practitioners on the specialities, rather than for general surgery. A number of distinguished contributors have brought the contents up to date. A particularly useful section is that on orthopaedic surgery.

This volume should continue to be popular with its valuable content, easy reference, and clear presentation. P.A.T.

CORRESPONDENCE : BRIEWERUBRIEK

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D. du Plessis

Mediese Sentrum 626
Pretoriusstraat
Pretoria
16 November 1960

43rd MEDICAL CONGRESS (M.A.S.A.), CAPE TOWN, 24 – 30 SEPTEMBER 1961 : 43ste MEDIESE KONGRES (M.V.S.A.), KAAPSTAD, 24 - 30 SEPTEMBER 1961

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